Public Health Reports

VOLUME 58 JANUARY 1, 1943 NUMBER 1

IN THIS ISSUE

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Public Health Reports

Vol. 58 • JANUARY 1, 1943 • *No. 1*

COCCIDIOIDOMYCOSIS IN WILD RODENTS. A METHOD OF DETERMINING THE EXTENT OF ENDEMIC AREAS [1]

By C. W. EMMONS, *Principal Mycologist, United States Public Health Service*

The occurrence of coccidioidomycosis in wild rodents has been previously reported (*3, 6, 7*). The character of the lesions and the prevalence of the disease in some species of rodents, in contrast to its infrequent occurrence in other species, was interpreted to indicate that certain wild animals living in areas where the disease is endemic constitute a natural reservoir. The animals collected in these earlier studies were trapped in the vicinity of San Carlos, Ariz. None of the trapping areas were in proximity to human dwellings.

The purpose of this paper is to report that coccidioidomycosis has now been found in rodents in other areas. This finding establishes the fact that the occurrence of the disease in rodents is not an isolated phenomenon; it further supports the concept that rodents constitute a reservoir of importance in the epidemiology of the disease; and it suggests a method of determining the geographical extent of endemic areas.

The data to be reported were obtained from a rapid survey [2] of the rodent population made in five areas in New Mexico and Arizona. These areas and the species of rodents trapped in each are listed in table 1. Previously recognized human cases of coccidioidomycosis or evidence from skin testing had already indicated that the Tucson, Casa Grande, and Phoenix areas were within the endemic localities of the disease (*2, 8, 10*). No such evidence was available from the Lordsburg, N. Mex., and Wilcox, Ariz., areas. Between September 3 and October 9, 1942, 1,942 rodents were trapped and examined. Of these, 312 were caught in box traps and killed just before examination. The remainder were caught in snap traps and, with few exceptions, were dead when found. Cultures were made from 207 animals, in most cases in triplicate. Most of these were made from those

[1] From the Division of Infectious Diseases, National Institute of Health.

[2] The investigation was aided by Medical Director N. E. Wayson, Medical Officer in Charge, Plague Suppressive Measures, U. S. Public Health Service, San Francisco, who permitted the temporary diversion for the purposes of this study of one of the mobile laboratory units of that office. The unit was operated by Dicran V. Turkian and John W. Anderson, who assisted with the mycological studies while carrying on the usual routine examinations for plague.

species of rodents which previous experience had shown were most likely to have mycotic infections. Cultures were made in the open with little protection from wind and dust, and the number of contaminated cultures was high. On a few days weather conditions were so unfavorable that no cultures were made. For these reasons the data obtained are qualitative rather than quantitative.

TABLE 1.—*Animals trapped, tabulated according to species and trapping area*

	Lordsburg	Wilcox	Tucson	Casa Grande	Phoenix	Total
Perognathus baileyi		12	80	5	3	100
P. intermedius and P. penicillatus	16	21	74	70	42	223
P. apache			2	9		11
P. flavus	3			4		7
Dipodomys merriami	117	52	142	223	114	648
D. ordii	39	13				52
D. spectabilis	21	20		13	2	56
Onychomys leucogaster	84	54				138
O. torridus		2	22	21	11	56
Peromyscus eremicus	32	34	11	50	10	137
P. maniculatus	40	21	1			62
Neotoma albigula	166	112	30	9	4	321
Citellus harrisi	3	2	21	6	4	36
C. spilosoma	30	9				39
C. tereticaudus			43	7		50
Sylvilagus auduboni	2			1	1	4
Reithrodontomys megalotis	2					2
Total	555	352	426	418	191	1,942

As in the previous studies (*6*, *7*) infection was determined by observing typical lesions in the lung or by isolating a fungus in culture from the lung. The latter is a more productive method since many animals without grossly visible lesions yield positive cultures, and the findings by cultural methods can be confirmed by subsequent microscopic examination of tissue sections. Five infected animals were collected in the Wilcox area, and 20 in the Tucson, 21 in the Casa Grande, and 12 in the Phoenix areas (table 2). *Coccidioides immitis* was isolated from 3 animals at Tucson, 3 at Casa Grande, and 1 at Phoenix. From the other infected animals *Haplosporangium parvum* (*7*) was isolated, except that in 3 cases typical lesions were seen but no pathogen was obtained in pure culture. *C. immitis* was isolated from specimens of *Perognathus intermedius*, *P. penicillatus*, *P. baileyi*, and *Dipodomys merriami*. *H. parvum* was obtained from specimens of *P. intermedius*, *P. penicillatus*, *P. baileyi*, *D. merriami*, *Onychomys leucogaster*, and *O. torridus*. As in previous series, species of Perognathus were most important as hosts.

Fewer grossly visible lesions were observed in these animals than in those collected previously. While this may represent an actual geographical difference it seems much more probable that it was due to other factors, possibly to the age of the animals. A few of the animals in the present series were of immature size. Many others, although they had reached approximately adult size, exhibited

pelage markings or other characteristics indicating immaturity. It seems probable that a large proportion of the animals were not more than a few weeks or a few months old. Animals in which no lesions were seen, but from which fungi were isolated, might have developed granulomas had they lived for a longer interval after infection. Earlier studies (*3*) have indicated that the disease progresses slowly in natural infections in these animals.

TABLE 2—*Animals tabulated according to trapping area and identity of fungus isolated in culture*

	Lordsburg	Wilcox	Tucson	Casa Grande	Phoenix	Total
Number of animals trapped	555	352	426	418	191	1,942
Number of animals examined by culture	36	19	53	59	40	207
Animals from which *C. immitis* was isolated	0	0	3	3	1	7
Animals from which *H. parvum* was isolated	0	4	16	17	11	50
Animals with typical lesions but no pathogen isolated	0	1	1	1	0	3

Although the numbers of infected animals are not great the data indicate clearly that coccidioidomycosis is present in the Tucson, Casa Grande, and Phoenix areas, and that it probably is not present in the Lordsburg area. The evidence for the Wilcox area is not so clear, but the presence of *H. parvum* in rodents caught near Wilcox is interpreted, for reasons enumerated below, to mean that Coccidioides is probably present there also.

The two fungi have been found closely associated in previous studies. *Haplosporangium parvum*, first isolated from rodents near San Carlos, was described in an earlier publication (*7*). As explained in that report, it causes microscopic lung lesions in wild rodents and in experimentally infected white mice; it appears to be associated in wild rodents in some cases with granulomas indistinguishable from those caused by *C. immitis;* it is found in those species of rodents naturally infected with *C. immitis* but rarely or not at all in other closely associated species; and a skin testing material prepared from it appears to have an antigen in common with coccidioidin. It seems evident that there is some relationship between *H. parvum* and *C. immitis*. For the present, therefore, the isolation of *H. parvum* from the rodents of a given area is taken to indicate that *C. immitis* is probably also present in that area. The small number of cultures made may explain the failure to isolate *C. immitis* from the Wilcox area.

Evidence from epidemiological studies and case histories shows that the spores of Coccidioides must be present in windblown dust (*4, 5, 9*). The presence of coccidioidomycosis in rodents might be explained by assuming, as earlier theories have held, that the fungus

grows in soil, and that susceptible rodents, because of their intimate contact with soil, inevitably become infected. It seems more probable, for reasons given elsewhere (*6*), that the disease is primarily a rodent disease, transmitted frequently but accidentally to man through the medium of soil contaminated by rodents. It must be remembered that so far as is known coccidioidomycosis is not normally transmitted from man to man, and that no evidence has been presented that it can become established in a new area by the migration of infected persons. Other factors—perhaps climate, perhaps the presence of suitable animal species as hosts—are apparently necessary. Otherwise the disease must long ago have been spread far beyond the geographical limits of the San Joaquin Valley and the Southwest. It may be significant that species of Perognathus and Dipodomys in which coccidioidomycosis has been most frequently found range only in southwestern United States and northern Mexico (*1*).

Although it has not been finally determined whether *C. immitis* is primarily a soil inhabiting saprophyte or primarily a pathogen of rodents, it can be pointed out that in either case a sampling of the rodent population of an area offers a quick and dependable method of determining whether Coccidioides is present. This is an important consideration in the choice of an area into which a large susceptible human population is to be moved. Large numbers of such susceptible individuals are now concentrated in some of the areas mentioned above. While cases of clinical importance appear to be rare in the native human population of endemic areas, numerous cases of the disease must be expected when adults are brought in from nonendemic areas. Some will be of such severity as to entail hospitalization and considerable loss of time, and it is probable that a small percentage will develop the chronic type of the disease, coccidioidal granuloma.

Two methods of determining the presence of coccidioidomycosis in an area are available. Persons can be skin tested with coccidioidin, and samples of the rodent population can be examined by the laboratory methods just outlined. Certain disadvantages inherent in the first method decrease its accuracy in tracing the precise limits within which the fungus is established. The human population shifts, and the exposure experienced during a few hours spent in an endemic area can be sufficient to evoke skin sensitivity (*9*). The use of coccidioidin necessitates the skin testing of a large number of people, and these individuals should be young children or others who have never traveled outside the area under investigation. The rodent population, on the other hand, is localized and adequate samples can be easily taken. The presence of *C. immitis* in a given area would appear to be more easily and clearly proved by its actual demonstration in cultures made from the lungs of native wild rodents than by the demonstration of coccidioidin sensitivity in the human population.

SUMMARY

Coccidioidomycosis was found in wild rodents in additional localities in Arizona. It was not found in an area examined in New Mexico. Rodents appear to constitute a natural reservoir of the disease, and the presence of susceptible species may explain the endemicity of coccidioidomycosis. It is suggested that examination and culture of the lungs from samples of the rodent population (particularly of species of Perognathus) offer a quick and dependable method of determining whether Coccidioides is present in a specific locality. This information may be of value in deciding whether unnecessary risk is involved in the concentration in certain areas of individuals from nonendemic areas.

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THE BACTERIOSTATIC ACTION OF SULFONAMIDE COMPOUNDS UPON CLOSTRIDIA [1]

By SANFORD M. ROSENTHAL, *Principal Pharmacologist, United States Public Health Service*

The use of the sulfonamides in the treatment of gas gangrene has been subjected to considerable investigation. The experimental approach has given conflicting results, but the most promising have been brought about by local application of the drugs (*1*, *2*). With this type of therapy it would seem that the effect of the drugs in the test tube might yield information of particular significance concerning

[1] From the Division of Chemotherapy, National Institute of Health.

their action in infected wounds. Therefore a series of sulfonamide and related compounds has been tested on six types of clostridia in an effort to determine their relative bacteriostatic power.

REVIEW OF THE LITERATURE

Spray (*3*) investigated the effects of sulfanilamide, sulfanilyl sulfanilamide, and prontosil upon various clostridia grown in dextrose peptone water. Sulfanilamide in concentrations of 0.2 to 0.05 percent was bacteriostatic for *Cl. tetani*, *novyi*, *septicum*, and *histolyticum*, but little effect was found upon *Cl. welchii*, *sporogenes*, *bifermenteres* or *botulinum A*. Burton, McLeod, McLeod, and Mayr-Harting (*4*) studied sulfapyridine, sulfanilamide, and some oxidation products of sulfanilamide. Sulfanilamide and sulfapyridine were inactive, both in broth and in an agar medium, against *Cl. welchii*, *septicum*, *novyi*, and *sporogenes*. 4-Nitrobenzene sulfonamide was inhibitory in concentrations of 0.005 to 0.002 percent. Gordon and McLeod (*5*) later employed sulfanilamide, sulfapyridine, and 4-nitrobenzene sulfonamide in the therapy of experimental mouse infections. Little effect from any of them was observed against *Cl. septicum*, *Cl. novyi*, or *Cl. welchii*.

EXPERIMENTAL

The following organisms were selected for study: *Cl. welchii*, *Cl. tetani*, *Cl. septicum*, *Cl. novyi*, *Cl. histolyticum*, and *Cl. sporogenes*.[2] A virulent strain of B-hemolytic streptococcus was included for comparison. The medium employed was beef infusion broth containing 2 percent neopeptone and 0.1 percent agar. The presence or absence of 0.2 percent dextrose did not appear to influence the bacteriostatic titres obtained with the drugs.

The tubes were placed in boiling water for 30 minutes and cooled immediately prior to inoculation. No vaseline seal was employed. The organisms were transferred from 18-hour cultures made in broth containing whole meat powder. The inocula consisted of approximately 2,000 bacteria. Concentrations of the drugs were begun at 0.1 percent except where insolubility precluded this concentration.

RESULTS

Amino compounds.—It is seen from table 1 that sulfanilamide, sulfapyridine, sulfathiazol, sulfadiazine, and 4,4′-diaminodiphenylsulfone were inactive or feebly active against all of the clostridia studied with one exception. *Cl. septicum* was inhibited by concentrations of 0.01 to 0.002 percent. In order to determine if this was a peculiarity of the strain employed, tests were repeated on two

[2] Obtained from Associate Bacteriologist Sarah E. Stewart of the National Institute of Health.

TABLE 1.—*The bacteriostatic action of sulfonamides against six species of clostridia*

	Cl. welchii		*Cl. tetani*		*Cl. septicum*		*Cl. novyi*		*Cl. histolyticum*		*Cl. sporogenes*		*Hemolytic streptococcus*	
	16 hours	24 hours	16 hours	24 hours	16 hours	24 hours	16 hours	24 hours	16 hours	24 hours	16 hours	24 hours	16 hours	24 hours
S. A.	P 1,000	0 1,000	0 1,000	0 1,000	P 10,000	P 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	P 1,000	0 1,000
4-Nitro S. A.	C 50,000	P 50,000	P 100,000	C 10,000	C 10,000	C 10,000	P 10,000	C 10,000	P 10,000	0 10,000	C 10,000	C 10,000	P 1,000	P 1,000
S. P.	P 1,000	P 1,000	0 1,000	0 1,000	C 10,000	P 1,000	0 1,000	0 1,000	P 1,000	0 1,000	0 1,000	0 1,000	P 1,000	P 1,000
S. D.	P 1,000	P 1,000	0 1,000	0 1,000	C 10,000	P 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	P 1,000	0 1,000
S. T.	P 1,000	P 1,000	0 1,000	0 1,000	C 10,000	P 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	P 10,000	P 1,000
4-Nitro S. T.	C 10,000	C 10,000	C 10,000	P 10,000	C 50,000	C 5,000	C 50,000	P 1,000	C 5,000	0 5,000	P 1,000	0 1,000	0 5,000	0 5,000
Di-amino sulfone	0 1,000	0 1,000	0 5,000	0 5,000	C 50,000	P 5,000	0 5,000	0 5,000	0 5,000	0 5,000	0 5,000	0 5,000	P 5,000	0 5,000
Nitro Amino Sulfone	C 10,000	C 10,000	P 100,000	P 50,000	C 50,000	C 50,000	C 50,000	P 25,000	P 5,000	P 5,000	P 5,000	0 5,000	P 5,000	0 5,000
4-Nitro S. A.+P. A. B.	C 10,000	C 10,000	C 10,000	C 10,000	C 5,000	C 5,000	C 5,000	C 5,000	P 10,000	0 10,000	C 10,000	0 10,000	P 1,000	P 1,000
S. T.+P. A. B.	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000	0 1,000

Figures represent dilution of one part of drug.
0 = No inhibition.
P = Partial inhibition.
C = Complete inhibition.
S. A. = Sulfanilamide.
S. P. = Sulfapyridine.
S. D. = Sulfadiazine.
S. T. = Sulfathiazol.
P. A. B. = Para-aminobenzoic acid.

additional strains of this organism with essentially similar results. With this exception, these compounds were less active against clostridia than upon a hemolytic streptococcus grown in the same medium.

Nitro compounds.—The findings of Burton et al. on the bacteriostatic power of 4-nitrobenzene sulfonamide against clostridia have been confirmed. Inhibition was obtained in concentrations of 0.01 to 0.001 percent. The nitro derivatives of sulfathiazol, sulfapyridine [3] (not shown in the table), and of 4-nitro-4′-aminodiphenylsulfone were also active in concentrations of 0.02 to 0.002 percent. In contrast to the effect on clostridia, the nitro compounds were of no greater activity than the amino compounds when tested upon the streptococcus.

To investigate the nature of the bacteriostatic action of these nitro compounds, the antagonism of 4-aminobenzoic acid was tested. No antagonism was observed upon either clostridia or streptococcus. With the amino compounds, when inhibition was obtained, it could be abolished by 4-aminobenzoic acid.

Further tests were done upon *Cl. welchii* with the following compounds. The lowest concentration of the drug which produced inhibition in 16 hours is shown in parenthesis (0=no inhibition, P=partial, C=complete):

3-Nitrobenzene sulfonamide (C-50,000)
Sulfanilamido-4-nitro-aniline (P-10,000)
2-Nitrobenzoic acid (P-1,000)
3-Nitrobenzoic acid (P-1,000)
4-Nitrobenzoic acid (P-1,000)
4-Nitrosulfanilic acid (P-1,000)
4-Hydroxy-4′-aminodiphenylsulfone (C-1,000)
Sulfanilic acid (0-1,000)

It is seen that the action of para-nitrobenzene sulfonamide differs from sulfanilamide in the lack of antagonism by para-amino benzoic acid and in the fact that the meta isomer is equally active. Changes in or replacements of the sulfonamide group affect activity to a marked extent.

RELATIONSHIP OF THE BACTERIOSTATIC ACTIVITY TO OXIDATION-REDUCTION POTENTIAL

Experiments were carried out in the sterile medium to determine the effects of the drugs on the redox potential of the medium. Concentrations of methylene blue and indigo disulfonate (1/10,000 molar) were employed as indicators. Conditions were kept similar to those employed in the bacteriostatic tests, and the degree of reduction was estimated after incubation at 37° C. for 16 to 24 hours. The hydrogen ion concentration of the medium, determined with the

[3] Obtained through the courtesy of Dr. Charles L. Fox.

glass electrode, was pH 7.1. In these experiments glucose was omitted from the medium.

Except for zones in contact with air, the medium alone was capable of reducing both indicators. Sulfanilamide in concentrations of 0.1 percent or less did not affect the rate of reduction of either dye. 4-Nitrobenzene sulfonamide inhibited the rate of reduction of methylene blue in concentrations of 0.1 percent, but not in lower concentrations. Reduction of indigo disulfonate by the medium was completely inhibited by the nitro compound in concentrations of 0.1 to 0.01 percent, and partially between 0.002 to 0.0004 percent. It is thus seen that 4-nitrobenzene sulfonamide in concentrations comparable to those causing bacteriostasis can elevate the redox potential of the medium.

SUMMARY

The commonly employed sulfonamide compounds showed little bacteriostatic action against five of six species of clostridia. Only *Cl. septicum* showed appreciable inhibition by compounds of this class.

Nitro derivatives of sulfanilamide, sulfathiazol, sulfapyridine, and 4,4′-diaminodiphenylsulfone were many times more active against these clostridia, although not more active against a hemolytic streptococcus.

The mechanism of action in the nitro compounds differs from that of sulfanilamide in that no antagonism is obtained with p-aminobenzoic acid. Also different from sulfanilamide is an elevation of the redox potential of the medium.

Further study seems indicated to obtain sulfonamides more active against clostridia infections.

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TREATMENT OF YOUNG WHITE MICE INFECTED WITH *LEPTOSPIRA ICTEROHAEMORRHAGIAE* WITH IMMUNE SERUM [1]

By CARL L. LARSON, *Assistant Surgeon, United States Public Health Service*

Considerable debate has arisen over the value of convalescent or hyperimmune serum in the treatment of Weil's disease, but little experimental work has been done to clarify the subject. The experiments presented here were designed to test this question and to indicate the value of specific immune serum as a therapeutic agent among mice suffering from leptospirosis. Young white mice are extremely susceptible to infections with *L. icterohaemorrhagiae* (*1*), and infection in them may be inhibited by passive protection (*2*).

Bassett-Smith (*3*) administered immune serum early in the course of the disease to guinea pigs suffering from leptospirosis and found the mortality to be considerably decreased. Serum from a horse immunized against *L. icterohaemorrhagiae* protected guinea pigs against infection with this organism and when given on or before the fourth day of infection, mortality was reduced to 16 percent, but when given beyond this time mortality amounted to 54 percent (*4*).

The strains of *L. icterohaemorrhagiae* used in the experiments to be reported have been carried in this laboratory for a considerable length of time. They have been maintained by passage through generations of young white mice and have a high degree of virulence for such hosts.

Two specimens of serum from human cases convalescent from Weil's disease were studied. Specimen 157 was drawn from the patient 51 days after onset of symptoms and specimen 172 was obtained 28 days after onset. Both samples agglutinated *L. icterohaemorrhagiae* to a titer of 1:100,000.

Plasma, serum, and concentrated serum were obtained from domestic rabbits which were inoculated with living cultures of *L. icterohaemorrhagiae*. No agglutinins against leptospirae were observed in the serum of any of the rabbits used for production of serum prior to the beginning of the immunization process. An initial dose of 0.5 cc. of a 6-day culture of virulent organisms was injected intraperitoneally into a group of 12 normal rabbits. After a week intravenous injection of gradually increasing volumes of cultures every 3 to 4 days was instituted. This was carried on for a period of 103 days during which time a total of 145 cc. of culture was given intravenously. At the end of this period pooled serum from the animals agglutinated *L. icterohaemorrhagiae* to a titer of 1:10,000,000.

Plasma was obtained by drawing 25 cc. of blood from each animal and mixing with equal quantities of 2.5 percent sodium citrate. The

[1] From the Division of Infectious Diseases, National Institute of Health.

plasma was found to have an agglutination titer of 1:1,000,000 against *L. icterohaemorrhagiae*.

One week following the above bleeding 50 cc. of blood were obtained from each animal. The serum obtained from this bleeding agglutinated *L. icterohaemorrhagiae* to a titer of 1:10,000,000. A portion of the serum was precipitated with barium sulfate and the globulin fraction was pressed, freed from diffusible material by dialysis, and dissolved in a quantity of 0.85 percent sodium chloride sufficient to bring it up to one-tenth of the original volume of serum used. The titer of agglutinins increased approximately ten times, although the endpoint was not sharply defined. This material was considered to be 100 percent concentrated immune serum and further dilutions with salt solution were made to obtain 50 percent and 25 percent concentrated immune serum. Table 1 summarizes the serums and plasma used in these experiments.

TABLE 1.—*Summary of serums and plasma tested for therapeutic effect on murine leptospirosis*

Source	Type	Agglutination titer versus *L. icterohaemorrhagiae*
Human	Convalescent serum (157)	1:100,000
Do	Convalescent serum (172)	1:100,000
Rabbit	Normal serum	0
Do	Hyperimmune plasma	1:1,000,000
Do	Hyperimmune serum	1:10,000,000
Do	Concentrated hyperimmune serum	+1:10,000,000 ±1:100,000,000

The method of testing the therapeutic efficacy of the above materials included titration of the infective agent, titration of the specific protective antibodies in the specimen of serum or plasma to be tested, and inoculation of this material into infected mice at suitable intervals in order to observe the therapeutic effects. Mice suffering from leptospirosis were sacrificed soon after the onset of symptoms to provide the infective agent. It had been observed that septicemia was most marked at this time. The liver, spleen, kidneys, and heart were removed, weighed, ground, and made into a 10 percent suspension in 0.85 percent salt solution. This was diluted so that tenfold serial dilutions from 10^{-1} to 10^{-5} were obtained. Doses of 0.3 cc. of these various dilutions were injected into the peritoneal cavity of six mice in order to titrate the infectivity of the tissue suspension.

The serum to be used was observed to insure sterility and then diluted serially with salt solution to the desired endpoint. These various dilutions of serum were then mixed in equal parts with a 10^{-2} suspension of infective material and allowed to stand at room temperature for 1 to 2 hours when 0.6 cc. of the mixture was injected into

the peritoneal cavity of each of five mice for each dilution. This procedure determines the protective titer of the serum.

The desired number of mice were inoculated intraperitoneally with 0.3 cc. of a 10^{-2} suspension of infected tissue and were subsequently used to test the therapeutic value of the serum. Serum either whole or diluted was later injected in 0.3 cc. amounts intraperitoneally into such infected mice at stated intervals.

All mice are observed for a 2-week period following initial infection.

EXPERIMENTAL

The results of tests on plasma separated from the blood of rabbits immunized against *L. icterohaemorrhagiae* are shown in table 2. The infective agent killed all the mice inoculated with a 10^{-2} dilution and 50 percent of those given a 10^{-3} dilution of the material. The plasma protected mice against infection when 0.3 cc. of a 10^{-3} dilution was administered intraperitoneally.

TABLE 2.—*Effect of varying dilutions of immune rabbit plasma administered with an infective dose* [1] *of* L. icterohaemorrhagiae *in young mice*

Dilution of plasma	Dose (cc.)	Survivors at end of 4 days	Survivors at end of 14 days	Ratio of protection
10^{-2}	0.3	6	5	5/6
10^{-3}	.3	6	6	6/6
10^{-4}	.3	6	0	0/6
10^{-5}	.3	6	0	0/6

[1] Infective dose = 0.3 cc. of 10^{-2} suspension having 50 percent endpoint of 10^{-3}.

Plasma was then tested as a therapeutic agent being given in 0.3 cc. quantities intraperitoneally at intervals of 1, 24, 48, and 72 hours after the infective material had been injected into the test animals. Normal rabbit serum failed to influence the course of the disease in mice. The results obtained (table 3) indicate that hyperimmune plasma is effective in the treatment of leptospirosis in young mice for at least 72 hours after infection has been induced.

TABLE 3.—*Effect of whole immune rabbit plasma and normal rabbit serum administered at varying intervals after an infective dose* [1] *of* L. icterohaemorrhagiae *in young mice*

Type of serum or plasma	Dose (cc.)	Interval (hours) between infecting dose and administration of serum or plasma	Number of mice treated	Number of mice surviving	Percent survivors
Normal serum	0.3	24	24	1	4.2
Immune plasma	.3	1	23	23	100
Do	.3	24	24	20	83.3
Do	.3	48	23	21	91.3
Do	.3	72	24	24	100

[1] Infective dose = 0.3 cc. of 10^{-2} tissue suspension having 50 percent endpoint of 10^{-3}.

The next experiment was devised to determine whether or not the therapeutically effective fraction of hyperimmune serum could be concentrated in the globulin portion of the serum. The results are given in table 4. The serum from which the concentrated globulin was subsequently obtained contained sufficient antibodies to protect all infected mice when given 0.3 cc. of a 10^{-4} dilution intraperitoneally. Concentrated immune serum (100 percent) protected mice against infection in doses of 0.3 cc. of a 10^{-5} dilution, while 50 percent and 25 percent dilutions of concentrated immune serum gave somewhat lower results. It is apparent that protective antibodies against *L. icterohaemorrhagiae* are contained in the globulin fraction of immune serum and may be concentrated by suitable means.

TABLE 4.—*Effect of varying dilutions of immune rabbit serum and concentrated immune rabbit serum (100, 50, and 25 percent) administered simultaneously with an infective dose* [1] *of* L. icterohaemorrhagiae *in young mice*

Type of serum	Dilution of serum	Dose of serum (cc.)	Survivors at end of 4 days	Survivors at end of 14 days	Ratio of protection
Immune serum	10^{-4}	0.3	6	6	6/6
Do	10^{-5}	.3	6	1	1/6
Do	10^{-6}	.3	6	0	0/6
Concentrated immune serum (100 percent)	10^{-4}	.3	6	6	6/6
Do	10^{-5}	.3	6	5	5/6
Do	10^{-6}	.3	6	1	1/6
Concentrated immune serum (50 percent)	10^{-4}	.3	6	6	6/6
Do	10^{-5}	.3	6	3	3/6
Do	10^{-6}	.3	6	2	2/6
Concentrated immune serum (25 percent)	10^{-4}	.3	6	6	6/6
Do	10^{-5}	.3	6	2	2/6
Do	10^{-6}	.3	6	0	0/6

[1] Infective dose = 0.3 cc. of 10^{-2} tissue suspension having 50 percent endpoint of $10^{-2.6}$.

NOTE.—All mice given immune serum or concentrated immune serum of dilutions of 10^{-4}, 10^{-5}, and 10^{-6} survived.

Immune serum and the concentrated fraction of this serum were then tested on groups of 24 infected mice, 1, 3, 5, and 6 days after exposure to infection (table 5). Both materials tested were effective when administered on the third day. Only about 50 percent of the animals survived when treatment was instituted on the fifth day and none survived when the serum was withheld until the sixth day. As a control, normal rabbit serum was administered in 0.3 cc. doses intraperitoneally to 24 infected mice 24 hours after infection had been induced. All but one of the animals succumbed and presented typical signs and symptoms of leptospirosis.

The value of human convalescent serum in murine leptospirosis was next considered. Table 6 shows that neither of the human serums protected mice against *L. icterohaemorrhagiae* to the same extent as did immune or concentrated immune rabbit serum.

TABLE 5.—*Effect of whole immune rabbit serum and concentrated immune rabbit serum (100 percent) and normal rabbit serum administered at varying intervals after an infective dose*[1] *of* L. icterohaemorrhagiae *in young mice*

Type of serum	Dose of serum (cc).	Interval (days) between infecting dose and administration of serum	Number of mice treated	Number of mice surviving	Percent survivors
Normal serum	0.3	1	24	1	4.2
Immune serum	.3	1	24	22	91.7
Do	.3	3	24	24	100
Do	.3	5	24	10	41.7
Do	.3	6	17	0	0
Concentrated immune serum (100 percent)	.3	1	24	24	100
Do	.3	3	24	22	91.7
Do	.3	5	24	14	58.3
Do	.3	6	17	0	0

[1] Infective dose = 0.3 cc. of 10^{-4} tissue suspension having 50 percent endpoint of $10^{-2.8}$.

TABLE 6.—*Effect of varying dilutions of immune rabbit serum, concentrated immune rabbit serum (100 percent), and human convalescent serum administered simultaneously with an infective dose*[1] *of* L. icterohaemorrhagiae *in young mice*

Type of serum	Dilution of serum	Dose of serum (cc.)	Survivors at end of 4 days	Survivors at end of 14 days	Ratio of protection
Immune rabbit serum	10^{-3}	0.3	6	6	6/6
Do	10^{-4}	.3	6	6	6/6
Do	10^{-5}	.3	6	2	2/6
Do	10^{-6}	.3	6	0	0/6
Concentrated immune rabbit serum (100 per cent)	10^{-3}	.3	6	6	6/6
Do	10^{-4}	.3	6	6	6/6
Do	10^{-5}	.3	6	5	5/6
Do	10^{-6}	.3	6	2	2/6
Human convalescent serum (157)	10^{-3}	.3	6	6	6/6
Do	10^{-4}	.3	6	3	3/6
Do	10^{-5}	.3	6	0	0/6
Do	10^{-6}	.3	6	0	0/6
Human convalescent serum (172)	10^{-3}	.3	6	5	5/6
Do	10^{-4}	.3	6	0	0/6
Do	10^{-5}	.3	6	0	0/6
Do	10^{-6}	.3	6	0	0/6

[1] Infective dose = 0.3 cc. of 10^{-3} tissue suspension having 50% endpoint of $10^{-3.1}$.

NOTE.—All mice given immune serum, concentrated immune serum, or human convalescent serum of dilutions of 10^{-1} and 10^{-2} survived.

Normal serum and the other materials tested for their protective ability were tested on groups of 12 infected mice each, 4 days after administration of the infective dose. The normal rabbit serum did not reduce the mortality among the 12 animals to which it was given but no deaths resulted among any of the groups receiving serums from individuals or animals which contained protective antibodies. It is interesting to observe that in spite of the obvious disparity in protective antibody titer of the immune serums, there was no observed

difference in the therapeutic effect produced when given to mice 4 days after infection had occurred.

TABLE 7.—*Effect of immune rabbit serum, concentrated immune rabbit serum (100 percent), human convalescent serum, and normal rabbit serum when administered 4 days after an infective dose* [1] *of* L. icterohaemorrhagiae *in young mice*

Type of serum	Dose of serum (cc.)	Number of mice treated	Number of mice surviving	Percent survivors
Normal rabbit serum	0.1	12	0	0
Immune rabbit serum	.1	12	12	100
Concentrated immune rabbit serum (100 percent)	.1	12	12	100
Human convalescent serum (157)	.1	12	12	100
Human convalescent serum (172)	.1	12	12	100

[1] Infective dose = 0.3 cc. of 10^{-2} tissue suspension having 50 percent endpoint of $10^{-3.1}$.

There can be no doubt from the data presented that human convalescent serum and products obtained from rabbits hyperimmunized against *L. icterohaemorrhagiae* have a marked therapeutic effect upon leptospirosis in young white mice. The effects were most marked when serum or plasma was administered within 96 hours after the mice had been infected. Among 238 infected mice treated on or before the fourth day of infection, 228, or 95.7 percent, recovered, while only 24, or 50 percent, of 48 mice treated on the fifth day recovered. No recoveries were noted when treatment was instituted on the sixth day. Only two, or 3.3 percent, of 60 infected mice given normal rabbit serum failed to succumb to the disease.

SUMMARY

Serum from patients convalescent from Weil's disease and immune rabbit serum and plasma prevent the death of young white mice infected with *L. icterohaemorrhagiae*.

The effect of these materials is marked if administered on or before the fourth day after infection.

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HERPES SIMPLEX VIRUS RECOVERED FROM THE SPINAL FLUID OF A SUSPECTED CASE OF LYMPHOCYTIC CHORIOMENINGITIS [1]

By CHARLES ARMSTRONG, *Senior Surgeon, United States Public Health Service*

A strain of herpes simplex virus was isolated at the National Institute of Health from the spinal fluid of a suspected case of lymphocytic choriomeningitis which occurred at Gallinger Municipal Hospital, Washington, D. C. Blood drawn during the attack failed to protect mice against herpes infection, while a sample drawn later had acquired protective properties. Demonstrable antibodies against choriomeningitis were absent from all samples tested.

The history of the patient, as revealed by Gallinger Hospital records kindly supplied by Dr. L. K. Sweet, is as follows:

HISTORY OF CASE

G. T., colored male, 15 years of age, was admitted to Gallinger Municipal Hospital, Washington, D. C., December 4, 1940. He had been quite well until the morning of December 3, 1940, when he awoke with a severe headache; he was both nauseated and dizzy, but did not vomit. His headache continued, became more severe, and he developed a severe pain in his eyes. He was feverish and gradually became stuporous.

Physical findings.—The patient was a fairly well developed and well nourished Negro boy who was semi-stuporous and critically ill; temperature 100.4° F., pulse 100, respiration, 24. Blood pressure 140 mm. mercury, systolic; 75 mm. mercury, diastolic. There was slight opisthotonos and marked nuchal rigidity. The Kernig and Brudzinski signs were positive. The tendon reflexes were normal. Examination of the fundus oculi showed no lesions. No herpes or other significant physical abnormalities were noted.

Laboratory findings.—Blood—erythrocytes, 4.2 million; leucocytes, 14,100; polymorphonuclear neutrophiles, 74 percent (6 percent of which were band forms and 1 percent were younger forms); lymphocytes, 25 percent; basophiles, 1 percent. Urine—normal. Spinal fluid—950 cells, of which 96 percent were lymphocytes and 4 percent were polymorphonuclears. The total protein, 120 mg. percent; sugar, 77 mg. percent; chlorides, 677 mg. percent; colloidal gold curve, .0012221000; reaction to Kahn test, negative; culture, no growth.

Course.—The temperature gradually fell to normal over a period of 4 days. Coincident with this there was a slowing of the pulse rate and marked clinical improvement with disappearance of stupor and

[1] From the Division of Infectious Diseases, National Institute of Health.

evidence of meningeal irritation. On December 14, 1940, 10 days after admission, the spinal fluid contained 69 cells per cubic millimeter, of which 90 percent were lymphocytes and 10 percent polymorphonuclears. The total protein was 80 mg. percent, the colloidal gold curve was .0000000000. The patient continued to be symptom free. On December 21, 1940, the spinal fluid contained only 19 lymphocytes per cubic millimeter.

The patient was discharged in good condition on December 24, 1940. He was seen again on February 15, 1941, at which time he was in good health, without physical abnormalities. He had been free from untoward symptoms since his discharge from the hospital. Clinically the ailment was thought to be choriomeningitis. The recovery, however, was more prompt than usually has been the case with choriomeningitis patients displaying symptoms of corresponding severity.

Isolation of the virus.—A sample of spinal fluid drawn at Gallinger Municipal Hospital on the morning of December 4, 1940, the second day of illness, and another, together with a sample of whole blood, drawn on the afternoon of the same day, were submitted to the National Institute of Health for study. Both spinal fluid samples were sterile to culture on ordinary media. Groups of six mice were inoculated intracerebrally with 0.04 cc. of spinal fluid from each tap, and a group of six mice and one guinea pig were inoculated with a mixture of equal parts of whipped blood and spinal fluid from the afternoon tap. The mice were all from one lot of Swiss mice reared at the National Institute of Health.

No evidence of illness developed except in the group of mice inoculated from the primary spinal tap. Two of these mice died during the night of December 7, 1940, and were partially eaten by cage mates. On December 11 a third mouse developed symptoms resembling choriomeningitis. The brain of this animal was emulsified in saline and a 1:10 and a 1:500 suspension immediately inoculated into groups of five mice each. The mice receiving the heavier suspension all developed a roughened coat and tremors and all died on December 14 with their legs markedly extended backward. One of the mice which received the 1:500 suspension died December 15, and four died December 16; all had symptoms suggesting choriomeningitis.

NATURE OF THE INFECTIOUS AGENT

The fatal ailment in mice has been repeatedly conveyed by emulsions of infected mouse brains which were sterile to culture and in which no organisms were visible in variously stained, or in dark-field preparations.

Filterability.—The supernatant fluid from a centrifuged suspension of finely ground infectious mouse brain suspended in broth passed Berkefeld N. filters which withheld ordinary bacteria.

Resistance to glycerine.—The virus, in mouse brains, has been found to withstand suspension in glycerine (50 percent in saline pH 7.6 at +4° C.) up to 7 months. Survival for longer intervals has not yet been investigated.

Resistance to heat.—The infective agent maintained its virulence after exposure to 45° C. for 30 minutes but was found to be inactivated by exposure to 50° C. for the same interval.

Source of the infectious agent.—The infectious agent, apparently a virus, is believed to have been derived from the spinal fluid of the patient, G. T., since the mice employed in its isolation were from our own laboratory stock, many of which have been inoculated intracerebrally with various materials, including spinal fluids, but without a similar virus having been encountered. Strains of experimental herpes virus have never been in the new building at the National Institute of Health. Moreover, serum-virus protection tests in mice carried out with the patient's serum drawn on January 4, 1940, and on February 4, 1941 (table 1), showed, on repeated trials, a definite increase in demonstrable antibodies to herpes virus in the later drawn sample. Thus, a human origin for the virus seemed probable. Demonstrable antibodies against choriomeningitis virus were absent from all samples.

TABLE 1.—*Serum-virus protection tests employing serum from patient G. T. (two bleedings) and the G. T. strain of virus*

	First test (Jan. 4, 1941)			Repeat test (June 4, 1941)	
Undiluted serum (2 parts)	Virus dilution (1 part)	Day of death of mice after inoculation	Mice survived	Day of death of mice after inoculation	Mice survived
G. T. Early bleeding (Jan. 4, 1941)	1:25	5, 5, 8, 8	0	5, 5, 6, 6	0
	1:50	6,6,6,6	0	5, 6, 6, 8	0
	1:100	10, 12	2	1, 7, 8, 9	0
G. T. Late bleeding (Feb. 5, 1941)	1:25	7, 7, 11	1	6, 11, 17	1
	1:50	----------	4	13	3
	1:100	----------	4	9	3

CHARACTERISTICS OF THE VIRUS

Infectivity for mice.—Suspensions of infected mouse brains conveyed infection to approximately 50 percent of white mice when 0.03 cc. of a 1:50,000 suspension in saline was inoculated intracerebrally. Infection was brought about, but less readily, by rubbing the virus into the skin, or by subcutaneous, intraperitoneal, or intranasal inoculation, or when given by stomach tube.

Symptoms in mice.—Mice inoculated intracerebrally with 0.03 cc. of concentrated emulsion (1:20) of infected mouse brain developed

rough fur and appeared ill in from 48 to 72 hours. The ailment progressed rapidly and the hind legs tended to become spastic, causing a stilted gait. When such an animal was lifted by the tail it would usually quickly pass into a titanic spasm with both front and hind legs extended backward. These spasms are similar to those seen in mice inoculated intracerebrally with choriomeningitis virus, but come on more suddenly and it is more exceptional for mice to recover temporarily from them than is the case with choriomeningitis infection. Death usually occurs in from 3 to 6 days. Virus is readily recovered from the central nervous system but not from other organs. This picture is strikingly similar to that described by Andervont (*3*) for mice infected with herpes simplex virus. When the virus is rubbed on the scarified skin, or is given subcutaneously into a paw, the mice tend to develop flaccid paralysis of the inoculated limb in from 6 to 10 days. The paralysis usually spreads and death results in from 1 to 3 days after onset.

Susceptibility of other species.—Cotton rats and rabbits are highly susceptible, while rhesus monkeys are resistant.

Culture.—The virus was successfully carried through eight successive transfers on the chorio-allantois of the developing chick embryo.

Pathology in mice.—This account of the findings in mice is summarized from pathological examinations reported by Dr. J. H. Peers, research associate at the National Institute of Health.

Following intracerebral inoculation, a nonhemorrhagic type of meningitis develops. The exudate may contain fibrin, together with mononuclear, lymphoid, and polymorphonulcear cells. Foci of intensified meningeal reactions occur when pyknosis and karyorrhexis may be marked. Beneath these foci the process is often found to involve the underlying parenchyma where microglial proliferation is often present. Various degrees of perivascular infiltration occur and may accompany penetrative vessels into the parenchyma. Similar lesions may involve the cerebellum as well as other portions of the brain and may also occur in the cord.

Variously sized, ill-defined areas may be found where the white matter appears loose and stringy and where microgliosis and even definite necrosis which suggests demyelination may occur. The gray matter may also be involved.

When the virus is inoculated into a rear foot, neuritis marked by a cellular infiltration of the sciatic nerve may develop. There may be complete to partial obliteration of neurons from the lumbar cord and the white matter may appear badly disorganized in the cerebellum. Necrosis of Purkinje's cells has been observed.

The choroid plexus of one or more ventricles often shows usually slight lymphocytic infiltration, while the ependyma is thinned or even absent in spots.

VIRUS DIFFERENTIATION

The virus was readily differentiated from choriomeningitis virus by its host range, by the incubation period following its intracerebral inoculation into mice, by the pathology produced, and, to a less extent, by the symptomatology. Cross immunity tests in mice and the serum-virus protection tests further indicated that the two viruses were immunologically distinct.

The virus was also readily differentiated from the Lansing strain of poliomyelitis, the F. A. strain of encephalomyelitis of mice (Theiler), and from the murine virus of Jungeblut and Sanders.

IDENTIFICATION OF THE VIRUS

The virus in all particulars behaved like a strain of herpes simplex. Cross protection tests when carried out between this strain of virus (G. T.) and an established (H. F.) strain of herpes, secured through the courtesy of Dr. T. M. Rivers of the Rockefeller Institute of Medical Research, indicated immunological similarity (table 2).

TABLE 2.—*Cross neutralization tests employing G. T. virus and antiserum, and herpes virus (H. F.) and antiserum*

Undiluted serum (2 parts)	Virus dilution (1 part)	Day of death of mice after inoculation	Mice survived	Undiluted serum (2 parts)	Virus dilution (1 part)	Day of death of mice after inoculation	Mice survived
Anti G. T. (rabbit)	G. T.			Anti-herpes (H. F.) (rabbit)	G. T.		
	1:30	7, 7, 12	1		1:30	6, 8, 10, 15	0
	1:60	7, 9	2		1:60	10, 12, 13	1
	1:120	-------	4		1:120	10	3
Normal rabbit (control)	G. T.			Normal rabbit (control)	G. T.		
	1:30	3, 3, 3, 3	0		1:30	5, 5, 5, 7	0
	1:60	3, 3, 4, 4	0		1:60	5, 5, 6, 6	0
	1:120	3, 3, 3, 4	0		1:120	5, 5, 6, 6	0
Anti G. T. (rabbit)	H. F. herpes			Anti-herpes (H. F.) (rabbit)	H. F. herpes		
	1:30	5	3		1:30	5, 6, 8	1
	1:60	-------	4		1:60	8	3
	1:120	-------	4		1:120	9	3
Normal rabbit (control)	H. F. herpes			Normal rabbit (control)	H. F. herpes		
	1:30	2, 3, 4	1		1:30	4, 5, 6, 13	0
	1:60	3, 3, 3, 3	0		1:60	5, 5, 9, 9	0
	1:120	4, 4, 4, 5	0		1:120	5, 6, 7, 9	0

DISCUSSION

A strain of herpes simplex virus was isolated from the spinal fluid of a clinically suspected case of choriomeningitis of the meningeal type. Beyond the clinical resemblance, no evidence supporting the diagnosis was adduced, since choriomeningitis virus was not isolated from the spinal fluid and the patient upon recovery showed no demonstrable antibodies against this virus. On the other hand, following his recovery the patient did develop protective antibodies against the

recovered strain of herpes virus. The criteria usually relied upon to establish the etiological agent in virus infections are, therefore, fulfilled, and it seems possible that herpes simplex virus may, in rare instances, be the specific causative agent of a portion of those cases of lymphocytic or aseptic meningitis for which the etiology has not been established.

Herpes simplex is a widely distributed virus and has, on several occasions, been isolated from the spinal fluid of individuals presumably suffering from some other ailment such as encephalitis or syphilis (*4*, *5*, *6*, *8*), so that it was not possible to determine which, if any, of the symptoms were due to the presence of herpes virus. The virus is also reported to have been isolated occasionally from the spinal fluid of persons with herpes (*7*, *8*, *9*).

SUMMARY

A strain of herpes simplex virus was isolated from the spinal fluid of a patient with meningeal symptoms suggesting a mild case of choriomeningitis. Specific herpes neutralizing antibodies developed in the patient's serum following recovery.

No laboratory confirmation of infection with any other virus was elicited.

The evidence suggests that herpes simplex virus may, in rare instances, be the causative agent for a portion of those cases of aseptic or lymphocytic meningitis for which the cause is otherwise undetermined.

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DEATHS DURING WEEK ENDED DECEMBER 19, 1942

[From the Weekly Mortality Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Dec. 19, 1942	Corresponding week 1941
Data from 88 cities of the United States:		
Total deaths	9,478	8,728
Average for 3 prior years	8,642	
Total deaths, first 50 weeks of year	421,756	417,236
Deaths per 1,000 population, first 50 weeks of year, annual rate	11.8	11.7
Deaths under 1 year of age	674	525
Average for 3 prior years	501	
Deaths under 1 year of age, first 50 weeks of year	29,249	26,483
Data from industrial insurance companies:		
Policies in force	65,272,092	64,742,923
Number of death claims	12,006	12,503
Death claims per 1,000 policies in force, annual rate	9.6	10.1
Death claims per 1,000 policies, first 50 weeks of year, annual rate	9.1	9.4

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED DECEMBER 26, 1942

Summary

Reports for the week ended December 26, 1942, showed an increase in only one, smallpox, of the nine common communicable diseases listed in the following table, and only the reports of meningococcus meningitis were above the corresponding 5-year (1937–41) median. Cumulative figures for the 51 weeks of the year are above the comparable 5-year medians for only measles and meningococcus meningitis.

The reports of meningococcus meningitis for the week declined from a total of 103 to 92. The number for the corresponding week of 1941 is 37, which was also the comparable 5-year median number. Greatest numbers reported for the week were 11 cases in Oregon, 10 in Virginia, 8 each in Indiana and Maryland, and 6 each in Massachusetts, New York, and Pennsylvania. The cumulative figure for the 51 weeks of the current year is 3,582, about 29 percent more than the greatest number for the period since 1937 when the comparable number was 5,307.

There were 23 cases of smallpox reported for the current week, exclusive of an aggregate of 42 cases in Pennsylvania during November and December, 33 of which were at Lewistown and 9 in the vicinity of Lancaster. Of the reports for the current week 13 cases were in Ohio and 6 in Indiana.

Reports of poliomyelitis decreased from 60 to 36 cases for the current week, 10 of which were in California, 7 in Texas, and 4 in New York. The cumulative total for the 51 weeks of the year is 4,143 as compared with 9,051 in 1941, which was also the median number for the period in the past 5 years.

Only 2,290 cases of influenza were reported for the week, as compared with 2,414 for the preceding week and 5-year median of 2,587. Cumulative figures for the 51-week period are 105,727 for the current year, 290,164 for the 5-year median, and 520,153 for the period in 1941. Most of the cases in 1941 occurred in the first few weeks of the year.

Reports of measles decreased from 4,779 to 4,018 cases for the current week. The corresponding median number is 4,544. Greatest numbers for the week were 909 in Pennsylvania, 352 in Massachusetts, and 311 in Washington.

Other reports for the week include 12 cases of amebic, 65 bacillary, and 17 undefined dysentery; 3 cases of infectious encephalitis, 27 of tularemia, and 77 of typhus fever.

The death rate for the week in 88 large cities of the United States was 12.3 per 1,000 population, as compared with 13.2 for the preceding week and a 3-year (1939–41) average of 12.2. The cumulative rate for 51 weeks in 1942 is 11.8, and 11.6 in 1941.

Telegraphic morbidity reports from State health officers for the week ended Dec. 26, 1942, and comparison with corresponding week of 1941 and 5-year median

In these tables a zero indicates a definite report, while leaders imply that, although none were reported, cases may have occurred.

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended—		Median, 1937–41	Week ended—		Median, 1937–41	Week ended—		Median, 1937–41	Week ended—		Median, 1937–41
	Dec. 26, 1942	Dec. 27, 1941		Dec. 26, 1942	Dec. 27, 1941		Dec. 26, 1942	Dec. 27, 1941		Dec. 26, 1942	Dec. 27, 1941	
NEW ENG.												
Maine	1	0	0	1			13	192	35	5	0	0
New Hampshire	1	0	0				14	6	2	0	2	0
Vermont	0	0	0				159	4	25	0	0	0
Massachusetts	4	1	4				352	118	178	6	3	1
Rhode Island	0	2	0				1	26	3	0	0	0
Connecticut	1	0	1	2	1	1	211	76	67	2	2	1
MID. ATL.												
New York	19	11	20	[1] 10	[1] 10	[1] 14	243	344	395	6	5	3
New Jersey	6	12	9	12	17	6	38	38	38	4	3	0
Pennsylvania	7	15	26	3			909	533	533	6	1	5
E. NO. CEN.												
Ohio	8	7	17	7	14	9	46	47	47	1	0	1
Indiana	8	10	17	9	37	31	75	25	25	8	0	0
Illinois	8	24	33	7	6	19	46	36	36	0	0	0
Michigan [3]	11	4	5	1		1	45	38	253	1	2	0
Wisconsin	3	0	1	31	31	35	164	172	172	3	2	0
W. NO. CEN.												
Minnesota	2	2	2	1			2	107	31	1	0	0
Iowa	1	2	4		1	5	64	75	75	0	0	0
Missouri	4	10	10	3	5	16	6	27	11	1	0	1
North Dakota	2	1	1	24	17	17	0	133	16	0	0	0
South Dakota	0	3	3				147	0	1	0	0	0
Nebraska	3	0	0	5			87	4	4	0	0	0
Kansas	8	3	5	7	10	10	25	117	88	1	1	2
SO. ATL.												
Delaware	0	0	0				0	0	3	0	0	0
Maryland [2]	8	9	9	11	5	8	3	133	9	8	1	1
Dist. of Col	0	0	1	3		3	0	0	3	1	1	0
Virginia	12	32	32	383	260	111	12	103	77	10	3	1
West Virginia	4	6	9	16	17	17	6	92	16	3	0	2
North Carolina	4	14	21	2	1	6	3	212	212	2	0	1
South Carolina	3	12	5	204	203	236	3	45	7	1	1	1
Georgia	6	14	14	71	13	68	13	66	25	2	0	0
Florida	0	9	8	1	16	11	1	2	2	0	0	0
E. SO. CEN.												
Kentucky	3	4	7	18	1	16	58	32	32	0	0	3
Tennessee	6	11	7	56	61	61	13	73	55	0	0	1
Alabama	15	12	13	143	59	170	1	5	19	1	3	3
Mississippi [2]	5	8	5							0	0	0
W. SO. CEN.												
Arkansas	6	12	7	41	81	81	58	49	49	0	0	0
Louisiana	9	8	8	9		10	44	3	2	1	2	1
Oklahoma	8	16	16	94	120	120	103	113	9	0	0	0
Texas	21	51	50	823	1,254	597	16	296	77	2	2	2
MOUNTAIN												
Montana	0	0	0	15	6	6	26	41	14	0	0	0
Idaho	4	1	1	1		1	60	4	4	0	0	0
Wyoming	0	0	0	66	10	10	10	3	3	0	1	0
Colorado	8	5	11	34	69	69	27	59	59	3	0	0
New Mexico	0	0	2	3		2	3	6	16	0	0	0
Arizona	0	3	3	83	157	131	1	62	3	0	0	0
Utah [2]	0	0	0	43	27	27	261	48	38	1	0	0
Nevada	0	0					5	0		0	0	
PACIFIC												
Washington	0	1	2	4	1		311	17	19	1	0	0
Oregon	2	3	2	13	17	71	289	84	13	11	0	0
California	20	14	22	30	60	60	41	546	190	0	2	2
Total	241	352	499	2,290	2,587	2,587	4,018	4,212	4,544	92	37	37
51 weeks	15,234	16,620	23,589	105,727	520,153	290,164	500,072	860,850	372,517	3,582	1,992	1,992

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended Dec. 26, 1942, and comparison with corresponding week of 1941 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended—		Median 1937–41	Week ended—		Median 1937–41	Week ended—		Median 1937–41	Week ended—		Median 1937–41
	Dec. 26, 1942	Dec. 27, 1941		Dec. 26, 1942	Dec. 27, 1941		Dec. 26, 1942	Dec. 27, 1941		Dec. 26, 1942	Dec. 27, 1941	
NEW ENG.												
Maine	0	1	1	8	11	8	0	0	0	0	1	1
New Hampshire	0	0	0	15	9	8	0	0	0	0	1	0
Vermont	0	0	0	1	1	7	0	0	0	0	0	0
Massachusetts	0	0	0	238	196	144	0	0	0	1	3	1
Rhode Island	0	0	0	3	9	7	0	0	0	0	0	0
Connecticut	0	1	0	28	17	54	0	0	0	0	1	0
MID. ATL.												
New York	4	6	1	279	299	333	0	0	0	3	13	8
New Jersey	0	2	1	47	95	95	0	0	0	0	1	1
Pennsylvania	0	4	1	157	180	276	14	0	0	0	6	9
E. NO. CEN.												
Ohio	0	2	1	243	225	231	13	0	1	0	7	3
Indiana	0	0	0	68	122	125	6	1	5	3	0	2
Illinois	0	2	1	168	182	326	1	1	3	4	7	1
Michigan [2]	0	0	0	95	160	294	0	0	2	0	6	2
Wisconsin	0	0	0	204	112	130	0	0	3	0	0	0
W. NO. CEN.												
Minnesota	1	4	1	56	58	58	0	5	17	3	0	0
Iowa	1	0	0	46	47	72	0	0	4	0	1	1
Missouri	0	0	0	66	78	81	0	0	1	5	0	4
North Dakota	1	0	0	14	5	13	0	1	1	0	0	0
South Dakota	0	1	1	23	37	17	2	0	2	0	0	0
Nebraska	0	0	0	20	24	16	0	0	1	0	0	0
Kansas	0	1	1	46	62	104	0	1	0	2	0	0
SO. ATL.												
Delaware	0	0	0	2	26	16	0	0	0	0	0	0
Maryland [2]	1	0	0	40	53	46	0	0	0	1	3	3
Dist. of Col	0	0	0	12	13	10	0	0	0	0	1	1
Virginia	1	2	1	45	49	35	0	0	0	3	8	4
West Virginia	0	0	1	37	57	61	0	0	0	0	2	1
North Carolina	0	0	0	39	26	40	0	0	0	0	0	1
South Carolina	0	1	0	11	7	10	0	0	0	2	0	0
Georgia	0	0	0	35	13	21	0	0	0	0	2	2
Florida	0	0	0	5	10	8	0	0	0	0	1	1
E. SO. CEN.												
Kentucky	1	2	0	22	48	54	1	0	0	1	2	2
Tennessee	2	2	0	58	70	70	0	0	0	1	1	1
Alabama	0	0	1	22	37	23	0	0	0	3	1	3
Mississippi [3]	2	1	1	2	6	6	0	0	0	1	0	0
W. SO. CEN												
Arkansas	1	0	0	4	6	8	0	1	1	1	0	0
Louisiana	0	0	0	4	8	8	0	0	0	4	9	5
Oklahoma	0	0	1	27	25	25	0	1	1	1	1	1
Texas	7	2	2	39	57	74	0	4	4	4	4	12
MOUNTAIN												
Montana	0	0	0	8	23	23	0	0	1	0	2	0
Idaho	[3] 1	0	0	4	8	8	0	0	1	0	0	0
Wyoming	0	0	0	46	4	4	0	0	0	0	0	0
Colorado	1	1	0	58	16	26	0	0	5	1	0	1
New Mexico	0	0	0	2	5	16	0	0	0	2	1	1
Arizona	2	0	0	3	9	7	0	1	0	2	0	0
Utah [2]	0	2	1	54	13	13	0	0	0	0	0	0
Nevada	0	0	-----	3	0	-----	0	0	-----	0	0	-----
PACIFIC												
Washington	0	1	0	9	26	48	0	1	0	0	0	0
Oregon	0	0	0	11	6	20	0	0	0	0	0	0
California	10	1	1	103	103	140	0	0	4	1	3	3
Total	36	39	36	2, 530	2, 651	3, 457	37	17	110	49	80	89
51 weeks	4, 143	9, 051	9, 051	123, 995	124, 813	158, 500	801	1, 331	9, 456	6, 652	8, 297	12, 630

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended Dec. 26, 1942, and comparison with corresponding week of 1941 and 5-year median—Con.

Division and State	Whooping cough		Week ended Dec. 26, 1942								
	Week ended—			Dysentery							
	Dec. 26, 1942	Dec. 28, 1941	Anthrax	Amebic	Bacillary	Unspecified	Encephalitis, infectious	Leprosy	Rocky Mt. spotted fever	Tularemia	Typhus fever
NEW ENG.											
Maine	39	19	0	0	0	0	0	0	0	0	0
New Hampshire	5	11	0	0	0	0	0	0	0	0	0
Vermont	42	17	0	0	0	0	0	0	0	0	0
Massachusetts	194	125	0	0	1	0	0	0	0	0	0
Rhode Island	26	26	0	0	0	0	0	0	0	0	0
Connecticut	29	38	0	0	0	0	0	0	0	0	0
MID. ATL.											
New York	321	392	0	1	17	0	1	0	0	0	1
New Jersey	130	146	0	1	0	0	0	0	0	0	0
Pennsylvania	253	139	0	0	0	0	0	0	0	2	0
E. NO. CEN.											
Ohio	133	161	0	0	1	0	0	0	0	3	0
Indiana	24	43	0	0	0	1	0	0	0	3	0
Illinois	89	177	0	1	1	0	0	0	0	2	0
Michigan [2]	191	163	0	0	1	0	0	0	0	0	0
Wisconsin	142	258	0	0	0	0	0	0	0	1	0
W. NO. CEN.											
Minnesota	30	30	0	3	0	0	0	0	0	0	0
Iowa	28	13	0	0	0	0	0	0	0	0	0
Missouri	11	19	0	0	0	0	1	0	0	0	0
North Dakota	7	2	0	0	0	0	0	0	0	0	0
South Dakota	1	1	0	0	0	0	0	0	0	0	0
Nebraska	1	2	0	0	0	0	0	0	0	0	0
Kansas	46	40	0	0	0	0	0	0	0	4	0
SO. ATL.											
Delaware	7	0	0	0	0	0	0	0	0	0	0
Maryland [2]	69	21	0	0	0	2	0	0	0	0	0
Dist. of Col.	9	11	0	0	0	0	0	0	0	0	0
Virginia	59	36	0	0	0	12	0	0	0	5	0
West Virginia	4	10	0	0	0	0	0	0	0	0	0
North Carolina	26	100	0	0	0	0	0	0	0	0	12
South Carolina	4	11	0	0	0	0	0	0	0	0	1
Georgia	13	1	0	3	1	0	0	0	0	1	17
Florida	5	11	0	0	0	0	0	0	0	0	3
E. SO. CEN.											
Kentucky	19	39	0	0	0	0	0	0	0	0	0
Tennessee	16	32	0	0	0	1	0	0	0	3	2
Alabama	43	21	0	0	0	0	0	0	0	0	12
Mississippi [2]			0	0	0	0	0	0	0	0	0
W. SO. CEN.											
Arkansas	26	10	0	1	1	0	0	0	0	1	0
Louisiana	0	1	0	0	0	0	0	0	0	1	1
Oklahoma	15	3	0	0	0	0	1	0	0	0	0
Texas	128	74	0	2	36	0	0	0	0	1	28
MOUNTAIN											
Montana	17	8	0	0	0	0	0	0	0	0	0
Idaho	1	1	0	0	0	0	0	0	1	0	0
Wyoming	6	5	0	0	0	0	0	0	0	0	0
Colorado	6	14	0	0	9	0	0	0	0	0	0
New Mexico	9	23	0	0	2	0	0	0	0	0	0
Arizona	0	51	0	0	0	0	0	0	0	0	0
Utah [2]	14	17	0	0	0	1	0	0	0	1	0
Nevada	0	0	0	0	0	0	0	0	0	0	0
PACIFIC											
Washington	16	55	0	0	0	0	0	0	0	0	0
Oregon	9	6	0	0	0	0	0	0	0	0	0
California	192	147	0	1	5	0	0	0	0	0	0
Total	2,455	2,530	0	13	66	17	3	0	1	28	77
51 weeks	175,244	205,011									

[1] New York City only.
[2] Period ended earlier than Saturday.
[4] Delayed report.

WEEKLY REPORTS FROM CITIES

City reports for week ended December 12, 1942

This table lists the reports from 78 cities of more than 10,000 population distributed throughout the United States, and represents a cross section of the current urban incidence of the diseases included in the table.

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
Baltimore, Md.	1	0	5	4	3	3	12	0	24	0	0	116
Barre, Vt.	0	0	.....	0	20	0	0	0	0	0	0	0
Billings, Mont.	0	0	.....	0	0	0	1	0	1	0	0	0
Birmingham, Ala.	1	0	8	2	0	1	4	0	2	0	0	0
Boise, Idaho	0	0	.....	0	0	0	0	0	0	0	0	0
Boston, Mass.	0	0	.....	0	0	0	21	0	0	0	0	0
Bridgeport, Conn.	0	0	.....	0	0	2	3	0	5	0	0	2
Brunswick, Ga.	0	0	.....	0	0	0	0	0	0	0	0	2
Buffalo, N. Y.	0	0	.....	1	53	1	3	0	11	0	0	31
Camden, N. J.	1	0	.....	0	2	0	2	0	3	0	0	1
Charleston, S. C.	0	0	37	1	0	0	0	0	2	0	0	0
Chicago, Ill.	7	0	4	2	51	1	32	0	65	0	1	75
Cincinnati, Ohio	1	0	1	0	9	0	7	0	19	0	0	7
Cleveland, Ohio	4	0	10	1	0	1	10	1	52	0	0	84
Columbus, Ohio	1	0	2	2	0	0	4	0	21	0	0	0
Concord, N. H.	0	0	.....	0	0	0	1	0	1	0	0	0
Cumberland, Md.	0	0	.....	0	0	0	1	0	0	0	0	0
Dallas, Texas	1	0	.....	0	0	0	2	0	4	0	1	9
Detroit, Mich.	2	0	.....	4	5	0	13	1	27	0	0	115
Duluth, Minn.	0	0	.....	0	0	0	0	0	1	0	0	1
Fall River, Mass.	1	0	.....	0	0	0	0	0	5	0	0	12
Fargo, N. Dak.	0	0	.....	0	0	0	0	0	0	0	0	0
Flint, Mich.	1	0	.....	0	0	0	1	0	2	0	0	12
Fort Wayne, Ind.	0	0	.....	0	0	0	4	0	0	0	0	0
Frederick, Md.	0	0	.....	0	0	0	0	0	0	0	0	0
Galveston, Texas	0	0	.....	0	0	0	0	0	0	0	0	0
Grand Rapids, Mich.	0	0	.....	0	0	0	2	0	2	0	0	5
Great Falls, Mont.	0	0	.....	0	3	0	1	0	4	0	0	0
Hartford, Conn.	0	0	.....	2	4	0	0	0	1	0	0	4
Helena, Mont.	0	0	.....	0	2	0	1	0	0	0	0	0
Houston, Texas	3	0	.....	0	1	0	8	0	2	0	1	0
Indianapolis, Ind.	2	0	.....	0	12	0	8	1	11	0	0	9
Kenosha, Wis.	0	0	.....	0	3	0	0	0	2	0	0	0
Little Rock, Ark.	0	0	6	0	0	0	3	0	0	0	0	0
Los Angeles, Calif.	4	0	12	1	8	0	7	5	37	0	0	20
Lynchburg, Va.	1	0	.....	0	0	0	2	0	4	0	0	0
Milwaukee, Wis.	0	0	1	1	42	0	2	0	58	0	0	27
Minneapolis, Minn.	1	0	.....	0	1	0	3	0	30	0	0	14
Missoula, Mont.	0	0	.....	0	0	0	0	0	0	0	0	0
Nashville, Tenn.	0	0	.....	2	0	0	5	0	0	0	0	0
Newark, N. J.	0	0	1	0	2	5	8	0	10	0	0	7
New Haven, Conn.	0	0	.....	0	0	0	2	0	3	0	0	4
New Orleans, La.	0	0	4	3	3	0	9	0	2	0	0	1
New York, N. Y.	20	0	16	4	8	8	64	0	138	0	4	80
Omaha, Nebr.	1	0	.....	0	0	0	4	0	2	0	0	0
Philadelphia, Pa.	4	0	3	1	464	5	29	0	48	0	0	117
Pittsburgh, Pa.	0	0	2	3	2	1	13	0	8	0	0	9
Portland, Maine	0	0	.....	0	0	1	2	1	3	0	0	50
Providence, R. I.	0	0	1	1	0	0	5	0	2	0	0	31
Racine, Wis.	0	0	.....	0	27	0	0	0	4	0	0	1
Reading, Pa.	0	0	1	0	15	0	3	0	0	0	0	8
Richmond, Va.	1	0	.....	0	1	4	4	0	3	0	0	2
Roanoke, Va.	0	0	.....	0	0	0	0	0	0	0	0	0
Rochester, N. Y.	0	0	.....	0	2	0	4	0	4	0	0	14
Saint Joseph, Mo.	0	0	.....	0	0	0	2	0	0	0	0	0
Saint Louis, Mo.	2	0	.....	0	2	1	16	0	12	0	0	5

WEEKLY REPORTS FROM CITIES

City reports for week ended December 12, 1942

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
Salt Lake City, Utah	1	0	------	0	178	0	3	0	19	0	0	6
San Antonio, Tex	3	0	2	2	0	0	9	2	1	0	0	4
San Francisco, Calif	0	0	1	0	11	1	10	0	8	0	0	14
Savannah, Ga	0	0	11	2	0	0	2	0	0	0	0	0
Seattle, Wash	0	0	------	1	15	0	5	0	3	0	0	15
Shreveport, La	0	0	------	1	0	0	6	0	0	0	0	0
South Bend, Ind	0	0	------	0	0	0	0	0	1	0	0	3
Spokane, Wash	0	0	------	0	34	0	2	0	3	0	0	0
Springfield, Ill	0	0	------	1	0	0	4	0	0	0	0	24
Springfield, Mass	0	0	------	0	7	0	5	0	81	0	0	5
Superior, Wis	1	0	------	0	2	0	0	0	1	0	0	12
Syracuse, N. Y	0	0	------	0	0	0	2	0	3	0	0	23
Takoma, Wash	0	0	------	0	69	0	3	0	1	0	0	4
Tampa, Fla	0	0	------	0	1	0	3	0	0	0	0	0
Terre Haute, Ind	0	0	------	1	0	0	2	0	1	0	0	0
Topeka, Kans	0	0	------	0	7	0	1	0	2	0	0	0
Trenton, N. J	0	0	4	1	1	0	3	0	6	0	0	0
Washington, D. C	3	0	7	1	3	0	13	0	14	0	0	17
Wheeling, W. Va	0	0	------	0	0	0	1	0	0	0	0	8
Wilmington, Del	0	0	------	1	0	0	1	0	0	0	0	3
Wilmington, N. C	2	0	------	0	0	0	3	0	1	0	0	3
Worcester, Mass	0	0	------	0	5	0	5	0	7	0	0	10

Anthrax.—Cases: Philadelphia, 1.
Dysentery, amebic.—Cases: Baltimore, 1; Los Angeles, 1; San Francisco, 2.
Dysentery, bacillary—Cases: Baltimore, 2; Buffalo, 5; Charleston, S. C., 10; Detroit, 2; Los Angeles, 4; New York, 2; Richmond, 2; Rochester, 2; St. Louis, 1.
Leprosy.—Cases: New Orleans, 1.
Tularemia.—Cases: Cleveland, 1.
Typhus fever.—Cases: Charleston, S. C., 2; Dallas, 1; Los Angeles, 1; Nashville, 1; San Antonio, 1; Savannah, 1.

Rates (annual basis) per 100,000 population for the group of 78 cities in the preceding table (estimated population, 1942, 31,935,700)

Period	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
		Cases	Deaths						
Week ended Dec. 12, 1942	11.43	22.70	7.84	173.56	67.27	128.50	0.00	1.14	167.36
Average, 1937–41	17.14	21.75	[1] 5.25	[2]176.67	[1] 57.43	144.04	1.15	3.63	171.56

[1] 3-year average, 1939-41.
[2] 5-year median.

PLAGUE INFECTION IN TACOMA, WASHINGTON

Under date of December 14, 1942, plague infection was reported proved in specimens of fleas and tissue from rats, *R. norvegicus*, taken in Tacoma, Washington, on December 2 and December 5 as follows: One specimen consisting of a pool of 125 fleas from 134 rats, one of

tissue from 1 rat, and two of tissue from lots of 4 and 27 rats, respectively.

TERRITORIES AND POSSESSIONS

Panama Canal Zone

Notifiable diseases—September 1942.—During the month of September 1942, certain notifiable diseases were reported in the Panama Canal Zone, and terminal cities, as follows:

Disease	Panama		Colon		Canal Zone		Outside the Zone and terminal cities		Total	
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
Chickenpox	25		7		1		2		35	
Diphtheria	17	1	3		11		4	1	35	2
Dysentery (amebic)	4		2	1	4		5		15	1
Encephalitis, lethargic		1								1
Malaria [1]	29		5		504		180	5	718	5
Measles	5				16		2		23	
Meningitis, meningococcus				1	1		1		2	1
Mumps	1				2				3	
Pneumonia		6		8	103	3		3	[2] 103	20
Relapsing fever	1								1	
Tuberculosis		14		3	10	1		7	[2] 10	25
Whooping cough				1	1				[2] 1	1

[1] Includes 193 recurrent cases.
[2] Cases reported in the Canal Zone only.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended November 28, 1942.—During the week ended November 28, 1942, cases of certain communicable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis		1		1	1	1				4
Chickenpox		19		277	448	77	139	29	70	1,059
Diphtheria		16	1	72	3	2	1			95
Dysentery				22			2			24
German measles				5	12		2		5	24
Influenza						5	4		16	25
Lethargic encephalitis						1				1
Measles				33	115	9	18		3	178
Mumps		26	2	66	613	46	60	43	246	1,102
Pneumonia		6			8	1			26	41
Poliomyelitis			2	1	2				2	7
Scarlet fever		3	15	132	96	16	21	36	57	376
Tuberculosis	2	11	4	123	57	24	6	33	22	282
Typhoid and paratyphoid fever				5	1					6
Undulant fever				1	2	1				4
Whooping cough		3	1	299	100	30	4	20	14	471
Other communicable diseases		1		8	228	50		1	7	295

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

NOTE.—Except in cases of unusual prevalence, only those places are included which had not previously reported any of the above-mentioned diseases, except yellow fever, during the current year. All reports of yellow fever are published currently.

A cumulative table showing the reported prevalence of these diseases for the year to date is published in the PUBLIC HEALTH REPORTS for the last Friday in each month.

(Few reports are available from the invaded countries of Europe and other nations in war zones.)

Plague

Indochina.—For the period November 11–20, 1942, 1 case of plague was reported in Indochina.

Smallpox

Ecuador.—Guayaquil and vicinity.—During the month of November 1942, 4 cases of smallpox with 1 death were reported in Guayaquil and vicinity, Ecuador.

Turkey.—During the week ended December 5, 1942, 142 cases of smallpox were reported in Turkey.

Typhus Fever

Hungary.—For the week ended November 28, 1942, 5 cases of typhus fever were reported in Hungary.

Rumania.—For the week ended November 28, 1942, 52 cases of typhus fever were reported in Rumania.

Turkey.—During the week ended December 5, 1942, 7 cases of typhus fever were reported in Turkey.

Yellow Fever

Venezuela—Bolivar State—Piar District—Municipality of Pedro Cova.—On August 13, 1942, 1 case of yellow fever occurred in the Municipality of Pedro Cova, Piar District, Bolivar State, Venezuela. The patient died on August 18, 1942. Immediate vaccinations were made among the inhabitants of the neighborhood.